DUAL FORMS OF PHASE IB ARRHYTHMIAS IN THE ANESTHETIZED DOG

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Signal averaging (20-30 beats)(SA) of orthogonal leads (X, Y, and Z leads) across the boundaries of the left anterior descending coronary artery (LAD) distribution was utilized to detect delayed activation and localized reentry in anesthetized dogs (N=33) subjected to LAD occlusion. Ventricular arrhythmias occurring 15 to 30 min after coronary artery occlusion (phase 1B) (N=23) were associated with SA delayed activation (145 ± 16 msec) within the LAD distribution exceeding that observed on the epicardial surface (96 ± 12 msec). The onset and offset of phase 1B ventricular arrhythmia was associated with the development and regression of SA delayed electrical activation. Two arrhythmia forms associated with SA delayed activation were observed during phase 1B. The first arrhythmia form (N=14) was closely coupled to the initiating beat (178 ± 15 msec) and was associated with continuous electrical activation bridging the interval between the sinus beat and the coupled ventricular extrasystole. The second arrhythmia form (N=18) was longer coupled, usually a fusion beat (332 ± 34 msec), and although associated with SA delayed electrical activation, had a mid-diastolic interval consistent with background noise. Close-coupled beats accounted for 29% and long-coupled beats accounting for 71% of phase 1B ventricular extrasystoles coupled to sinus beats. The data suggest that localized reentry can be demonstrated for some, but not all phase 1B arrhythmia. Although long coupled beats are associated with delayed activation in SA electrograms, a critical interval spanning the T wave demonstrates "electrical quiescence" using present SA techniques.

VENTRICULAR FIBRILLATION IS NOT AN ANODALLY INDUCED PHENOMENON IN OPEN-CHEST DOGS

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It is generally assumed that ventricular fibrillation (VF) evoked by electrical stimulation depends upon anodal excitation. This assumption is primarily based on experimental evidence that the ventricular vulnerable period appears coincidental with the period of maximal anodal supernormality (Cranefield et al., Am J Physiol 1957;190:383), and that with equal application of current near the VF threshold, 96% of all VF were of anodal origin (Harris and Moe, Am J Physiol 1942;136:318). However, in these studies, only a small number of recording electrodes were used, and determining the origin of activation after strong stimulation was sometimes difficult, probably due to technical limitations. To test the hypothesis that VF is an anodally induced phenomenon, 6 open-chest dogs were studied with computerized mapping techniques. A plaque electrode array containing 56 closely (2.5 to 5 mm) spaced bipolar electrodes was placed on RV. The baseline driving stimuli (S_1s) were given to the center and a 5 ms bipolar single premature stimulus (S_2) was given via two electrodes (one anodal and one cathodal) at the opposite edges of the plaque electrode array. With this arrangement, the anode and cathode were separated by recording electrodes, and the origin of excitation could be determined by isochronal activation maps. The strength-interval curves were determined for anodal and cathodal stimulation. The vulnerable period was scanned by S_2 of increasing strength to induce multiple responses (MR) or VF. Results: Compatible with previous investigations, a relatively supernormal period was observed only at the anodal site, and was coincidental with the most vulnerable period of the cardiac cycle. The strength-interval curve determined at the cathodal site was hyperboloid, rising smoothly while S_1-S_2 interval progressively decreased. Despite this difference, the origins of excitation at the onset of MR or VF could be anodal, cathodal, or both. When the S_2 polarity was reversed without changing the S_1-S_2 interval or the S_2 strength, the origin of six episodes of MR or VF stayed at the same site, and did not change according to the polarity of the S_2. These findings indicate that VF is not an anodally induced phenomenon. The preexisting electrophysiologic state at the site of stimulation determines the initiation and maintenance of MR or VF.

DISPERSION OF REPOLARIZATION INDUCED BY A NONUNIFORM SHOCK FIELD

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A dispersion of repolarization may contribute to arrhythmias. Since the ability of a strong shock during an action potential (shocked AP) to shorten subsequent paced AP's and prolong the shocked AP in frog ventricle depends on the shock (S2) strength, a nonuniform S2 may induce a dispersion of repolarization. To test this, we applied 5 ms rectangular S2 that had a nonuniform or uniform potential gradient during the AP of bathed frog ventricular strips. One group (n=6) had a partitioned bath to produce a nonuniform S2 of 40 V/cm in one half of the 1x6 mm strip (H) and <1 V/cm in the other half (L), and simultaneous intracellular AP recordings in H and L with glass microelectrodes positioned 1.4±0.4 mm apart (mean±sd). Another group (n=7) had uniform S2 and an AP recorded near the center of the strip. AP duration was determined at the time of maximum repolarization rate. S1 pacing at 0.5 Hz was performed at one end of the strip and conduction along the strip was monitored. S2 trials were repeated every 3-5 minutes at an S1-S2 interval of 300 ms. RESULTS: In both H and L, nonuniform S2 produced 1)cumulative shortening of paced AP's and 2)lengthening of each shocked AP compared with the paced AP preceding it. Uniform S2 of 1 V/cm did not shorten the paced AP's or lengthen the shocked AP's indicating that the AP changes in L were not due to the small potential gradient in L. Before beginning nonuniform S2 trials, AP duration was 601±72 ms in H and 602±71 ms in L (p=ns). During 15-20 trials, paced AP's were shortened to 490±51 ms in H and 515±39 ms in L while each shocked AP was lengthened, compared with the paced AP preceding it, to 636±40 ms in H and 561±21 ms in L (p<0.05). Therefore, paced AP's after shocks repolarized 25 ms earlier in H than in L and shocked AP's repolarized 75 ms later in H than in L. CONCLUSIONS: 1)Nonuniform shocks can induce repolarization time differences over a small distance. 2)During the prolonged AP in H, the AP in L is prolonged compared with the paced AP preceding it, possibly by intracellular current from H to L during phase 3. During the shortened AP in H, the reverse current may shorten the AP in L. Repolarization time differences and interactions induced by nonuniform gradients may play a role in defibrillation and postcardioversion arrhythmias.

LOW ENERGY ATRIAL DEFIBRILLATION IN A SHEEP MODEL OF ATRIAL FIBRILLATION.

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The efficacy and safety of internal atrial defibrillation (DFB) was evaluated in a sheep model of atrial fibrillation (AF). In 9 pentobarbital-anesthetized sheep, a RA spring electrode was inserted via the right external jugular vein and a cutaneous patch electrode was placed on the left chest wall. Sustained AF was induced by rapid RA pacing and terminated by biphasic cathodal DFB synchronized to the ECG R wave with an external programmable defibrillator. Initial energies were low (<0.4 joules(J)) and were increased until AF converted to sinus rhythm. Repeated DFBs were performed at 4-6 energy levels (max 5J) about each threshold in balanced random order to determine the DFB curve. RESULTS:

Energy (J)	Number of DFB	% Success	% VF
<0.5	73	34	0
0.5-<1.0	67	52	1.5
1.0-<2.0	122	53	1.6
2.0-3.0	88	77	1.1
5	33	70	12.1
Total:	383	56.4	2.1

CONCLUSIONS: In this model, low energy DFB from AF to sinus rhythm is feasible using a right atrium/cutaneous patch electrode configuration. The probability of successful DBF depends on energy used, and the risk of ventricular fibrillation increases with higher energy DFBs and failure of reliable R-wave detection.